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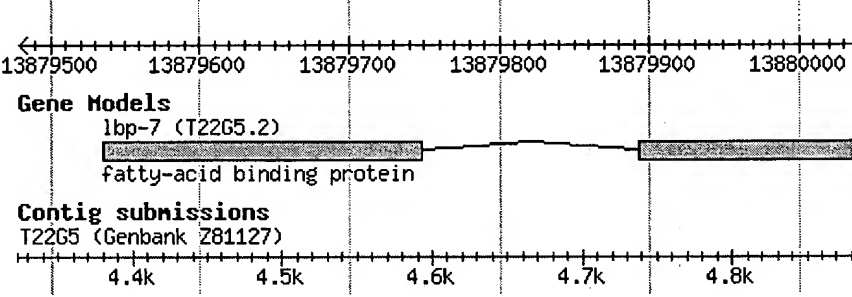
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Gene Summary for lbp-7

Specify a gene using a gene name (unc-26), a predicted gene id (R13A5.9), or a protein ID (CE02711) | lbp-7

[\[identification\]](#)
[\[location\]](#)
[\[function\]](#)
[\[expression\]](#)
[\[gene ontology\]](#)
[\[alleles\]](#)
[\[similarities\]](#)
[\[reagents\]](#)

Identification	IDs:	Main name	Sequence name	Other name(s)	W
		lbp-7 - (<i>Lipid Binding Protein</i>) via person evidence: John Plenefisch	T22G5.2	NM_074039 (inferred automatically) 5O359 (inferred automatically)	WBC
	Concise Description:	lbp-7 encodes a predicted intracellular fatty acid binding protein (i most similar to the vertebrate muscle and heart FABPs; by homol predicted to function as an intracellular transporter for small hydro molecules such as lipids and steroid hormones; as loss of lbp-7 a large-scale RNAi screens does not result in any obvious abnormal precise role of LBP-7 in C. elegans development and/or behavior known. [details]			
	NCBI KOGs*:	Fatty acid-binding protein FABP [KOG4015]; [OMpre_WH000485			
	Species:	<i>Caenorhabditis elegans</i>			
	NCBI:	[AceView: 5O359]			
	Gene model (s):	Gene Model	Status	Nucleotides (coding/transcript)	Protein
		T22G5.2 1	partially confirmed by cDNA(s)	414/559 bp	WP:CE1:
 Footnotes					

	History	
Location	<p>Interpolated V:5.72</p> <p>Genetic Position:</p> <p>Genomic Position:</p> <p>Genomic Environs:</p> 	
Function	<p>Pre-WormBase information: Definitions of abbreviations used in the text.</p> <p>Phenotypes: ☐ phenotypes via RNAi</p> <p>Interactions: ☐ Interactions</p> <p>Microarray Expression Data: SMD_T22G5.2 192878_at Aff_T22G5.2 cea2.i.45011</p> <p>Expression Cluster: cgc4386_cluster_1_3 cgc4489_group_1 WBPaper00024393:strongly_regulated_dau WBPaper00025040:N2_Expressed_Genes WBPaper00026980:intestine_enriched WBPaper00029437:rde-4_upregulated [cgc5976]:class_2 [cgc6390]:Cluster_C</p> <p>Microarray "topology map" position: Mountain [see Kim et al. Science (2001)]</p> <p>Regulation on Expression Level: nhr-49 [details]</p> <p>Protein domains: Calycin Lipocalin-related protein and Bos/Can/Equ α Cytosolic fatty-acid binding Lipocalin / cytosolic fatty-acid binding protein [more ...]</p>	
Gene Ontology	<p><i>lbp-7 gene ontology summary</i></p> <p>Biological process determination of adult life (IMP) via Phenotype: 0000039; span 00063219 transport (IEA) via InterPro</p>	

	<div>Molecular function</div> <div><div>binding</div><div>lipid binding</div><div>transporter activity</div></div> <div><div>(IEA) via InterPro</div><div>(IEA) via InterPro</div><div>(IEA) via InterPro</div></div>															
Alleles																
Homology	<div><div>Ortholog(s):</div><div><div>Caenorhabditis briggsae: Cbr-lbp-7 [syntenic alignment] (via a OrthoMCL; TreeFam; Inparanoid_6; OMA; WormBase-Compara)</div><div>Caenorhabditis briggsae: CBG11456 [syntenic alignment] (via a WormBase-Compara)</div><div>Caenorhabditis remanei: Cre-lbp-7 [syntenic alignment] (via a WormBase-Compara; Inparanoid_6; TreeFam)</div></div></div> <div><div>InParanoid group(s):</div><div><div>⊕ 2 InParanoid groups for lbp-7</div><div>Read more about InParanoid on the WormBaseWiki</div></div></div> <div><div>TreeFam:</div><div><div>TreeFam ID: TF316894</div><div>⊕ Treefam image</div><div>Phylogenetic trees provided by the Treefam project.</div></div></div>															
Similarities	<div><div>Best BLASTP matches to longest protein product (full list):</div><div>(Show alignments):</div><table><tr><th>Species</th><th>Hit</th><th>Description</th></tr><tr><td>C. remanei</td><td>RP03217 Note: C. remanei predictions are based on an early assembly of the genome. Predictions subject to possibly dramatic revision pending final assembly. Sequences available on the WormBase FTP site.</td><td></td></tr><tr><td>C. briggsae</td><td>BP:CBP02793</td><td>gene CBG11457</td></tr><tr><td>C. elegans</td><td>WP:CE41159</td><td>retinol binding protein</td></tr><tr><td>S. tridecemlineatus</td><td>SW:Q99P61</td><td>Fatty acid-binding protein, heart (H-FABP) (Heart-type fatty acid-</td></tr></table></div>	Species	Hit	Description	C. remanei	RP03217 Note: C. remanei predictions are based on an early assembly of the genome. Predictions subject to possibly dramatic revision pending final assembly. Sequences available on the WormBase FTP site.		C. briggsae	BP:CBP02793	gene CBG11457	C. elegans	WP:CE41159	retinol binding protein	S. tridecemlineatus	SW:Q99P61	Fatty acid-binding protein, heart (H-FABP) (Heart-type fatty acid-
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C. briggsae	BP:CBP02793	gene CBG11457														
C. elegans	WP:CE41159	retinol binding protein														
S. tridecemlineatus	SW:Q99P61	Fatty acid-binding protein, heart (H-FABP) (Heart-type fatty acid-														

				sbinding protein).
	C. hircus	TR:Q6S4N9		Fatty acid-binding protein.
	H. sapiens	ENSEMBL:ENSP00000362817		Fatty acid-binding protein, heart
	D. melanogaster	FLYBASE:CG6783-PB		Flybase gene name is CG6783-PB
Reagents	ORFeome Project primers & sequences: mv_T22G5.2 OSTF158E7_1 OSTR158E7_1 Primer pairs: sjj_T22G5.2 cenix:44-g12 Microarray probes: 192878_at cea2.i.45011 A_12_i [Affymetrix] [GSC] [Agilen] SAGE Tags: SAGE:gaactttgat SAGE:aaggaaatcg Matching cDNAs: OSTF158E7_1 OSTR158E7_1			
Bibliography	Total citations for lbp-7: 4 [view all]			

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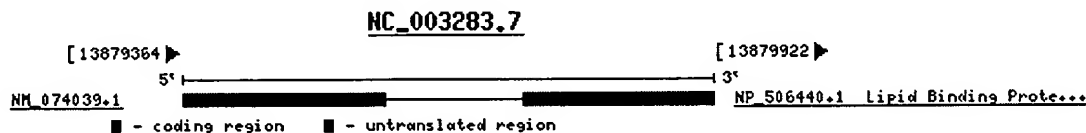
1: lbp-7 Lipid Binding Protein [*Caenorhabditis elegans*]

GeneID: 191701

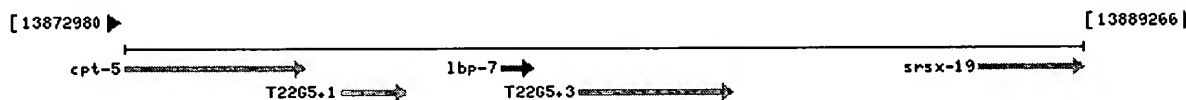
updated 13-Dec-2007

Summary

Gene name	lbp-7
Gene description	Lipid Binding Protein
Primary source	WormBase:WBGene00002259
Locus tag	T22G5.2
Gene type	protein coding
RefSeq status	Reviewed
Organism	<i>Caenorhabditis elegans</i> (strain: Bristol N2)
Lineage	Eukaryota; Metazoa; Nematoda; Chromadorea; Rhabditida; Rhabditoidea; Rhabditidae; Peloderinae; <i>Caenorhabditis</i>

Genomic regions, transcripts, and products[Go to reference sequence details](#)**Genomic context**

chromosome: V

[See lbp-7 in MapViewer](#)**Bibliography****Related Articles in PubMed**[PubMed links](#)[GeneRIFs: Gene References Into Function](#)**Interactions**

Description					
Product	Interactant	Other Gene	Complex	Source	Pubs
Two-hybrid					
BioGRID:56199	BioGRID:43789	W02G9.3		BioGRID	PubMed

General gene information

GeneOntology

Provided by WormBase

Function	Evidence
binding	IEA
PubMed 12520011,12654719	
lipid binding	IEA
PubMed 12520011,12654719	
transporter activity	IEA
PubMed 12520011,12654719	

Process	Evidence
determination of adult life span	IMP
PubMed 12845331	
transport	IEA
PubMed 12520011,12654719	

General protein information

Names

Lipid Binding Protein family member (lbp-7)

NCBI Reference Sequences (RefSeq)

*mRNA and Protein(s)*1. **NM_074039.1→NP_506440.1 Lipid Binding Protein family member (lbp-7)****[*Caenorhabditis elegans*]**

UniProtKB/Swiss-Prot O02323

Conserved Domains (1) summary**pfam00061**
Location:9-123
Blast Score:119

Lipocalin; Lipocalin / cytosolic fatty-acid binding protein family. Lipocalins are transporters for small hydrophobic molecules, such as lipids, steroid hormones, bilins, and retinoids. The family also encompasses the enzyme prostaglandin D synthase (EC:5.3.99.2).

Related Sequences

Nucleotide		Protein
Genomic	Z81127.1	CAB03387.1

Protein Accession	Links
O02323.1	GenPept

Additional Links

- UniGene Cel.2988

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L2 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2006:681667 BIOSIS <<LOGINID::20080104>>

DN PREV200600684105

TI Changes in the flexion relaxation response following an exercise intervention.

AU Marshall, Paul [Reprint Author]; Murphy, Bernadette

CS Univ Auckland, Dept Sport and Exercise Sci, Tamaki Campus, Private Bag 92019, Auckland, New Zealand
p.marshall@auckland.ac.nz

SO Spine, (NOV 1 2006) Vol. 31, No. 23, pp. E877-E883.

CODEN: SPINDD. ISSN: 0362-2436.

DT Article

LA English

ED Entered STN: 6 Dec 2006

Last Updated on STN: 6 Dec 2006

AB Study Design. Pre and post 12-week training study using surface electromyography to measure the flexion relaxation response. Objective. To evaluate whether the active or passive phases of the flexion relaxation measurement changes following an exercise intervention in patients with low back pain. Summary of Background Data. Impaired neuromuscular activation is an area of specific interest in patients with chronic nonspecific low back pain (LBP). The flexion relaxation phenomenon is commonly measured in LBP patients; however, there is insufficient evidence about the changes in this measure following an intervention. Methods. Fifteen subjects with chronic ***LBP*** (***7*** females, 8 males) performed a 12-week training intervention. The main outcome measures were the Oswestry disability index, visual analog scale, and flexion relaxation response analyzed by the raw electromyograph (EMG) signal, the relative EMG signal, and the flexion relaxation ratio. Results. Disability and pain scores improved significantly after the 12-week intervention. There were no changes in the active components of the flexion relaxation measurement but an approximate 67% decrease in the amount of activity measured during the relaxation phase at full trunk flexion. Conclusion. The data suggest that afferent feedback changes may be explaining why there is improved electrical relaxation following an exercise intervention.

L2 ANSWER 2 OF 4 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

AN 2006180752 EMBASE <<LOGINID::20080104>>

TI Plantar Infrared Thermography Measurements and Low Back Pain Intensity.

AU Zaproudina N.; Ming Z.; Hanninen O.O.P.

CS Dr. N. Zaproudina, Department of Physiology, University of Kuopio, Kuopio, Finland. nina.zaproudina@uku.fi

SO Journal of Manipulative and Physiological Therapeutics, (Mar 2006) Vol. 29, No. 3, pp. 219-223.

Refs: 19

ISSN: 0161-4754 CODEN: JMPTDE

PUI S 0161-4754(06)00025-X

CY United States

DT Journal; Article

FS 019 Rehabilitation and Physical Medicine

008 Neurology and Neurosurgery

LA English

SL English

ED Entered STN: 6 Jun 2006

Last Updated on STN: 6 Jun 2006

AB Objective: To study the skin temperature disorders in low back pain (LBP) patients compared with reference persons without LBP and to evaluate the relationship between pain intensity and other clinical signs and temperature abnormalities. Methods: Sixty-five patients with unilateral chronic LBP with or without referred nonradicular leg pain (29 men and 36 women; age range, 30-51 years) and 20 reference persons without ***LBP*** (***7*** men and 13 women; age range, 30-49 years) participated in this study. The pain level was recorded by the use of a visual analog scale (0-100). Questionnaires and a series of spinal mobility tests (the modified Schober, straight leg-raising test, finger-floor distance, side bending) were used. Thermographic images of the low back area and legs (anterior, lateral, and posterior surfaces and the plantar surfaces of feet) were taken with an infrared video camera. Results: The temperature changes in the plantar surface correlated with LBP intensity. The pain levels differed in the groups with the different types of temperature changes. There were significant lower extremity regional skin temperature alterations (at least 1 regional interside difference more than 0.3 degree.C) in most cases both in LBP patients and in reference persons, but plantar interside temperature difference was significantly higher in LBP patients. Conclusion: Temperature changes of the plantar surface seem to be connected with LBP intensity. Temperature

measurements may be useful as an adjunctive physiological test in the evaluation and documentation of autonomic dysfunction in LBP patients. .COPYRG. 2006 National University of Health Sciences.

L2 ANSWER 3 OF 4 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

AN 2006537243 EMBASE <<LOGINID::20080104>>

TI Changes in the flexion relaxation response following an exercise intervention.

AU Marshall P.; Murphy B.

CS P. Marshall, Department of Sport and Exercise Science, University of Auckland, Tamaki Campus, Private Bag 92019, Auckland, New Zealand. p.marshall@auckland.ac.nz

SO Spine, (Nov 2006) Vol. 31, No. 23, pp. E877-E883.

Refs: 45

ISSN: 0362-2436 E-ISSN: 1528-1159 CODEN: SPINDD

PUI 0000763220061101000025

CY United States

DT Journal; Article

FS 033 Orthopedic Surgery

005 General Pathology and Pathological Anatomy

008 Neurology and Neurosurgery

LA English

SL English

ED Entered STN: 16 Nov 2006

Last Updated on STN: 16 Nov 2006

AB STUDY DESIGN. Pre and post 12-week training study using surface electromyography to measure the flexion relaxation response. OBJECTIVE.

To evaluate whether the active or passive phases of the flexion relaxation measurement changes following an exercise intervention in patients with low back pain. SUMMARY OF BACKGROUND DATA. Impaired neuromuscular

activation is an area of specific interest in patients with chronic nonspecific low back pain (LBP). The flexion relaxation phenomenon is commonly measured in LBP patients; however, there is insufficient evidence about the changes in this measure following an intervention. METHODS. Fifteen subjects with chronic ***LBP*** (**** females, 8 males) performed a 12-week training intervention. The main outcome measures were the Oswestry disability index, visual analog scale, and flexion relaxation response analyzed by the raw electromyograph (EMG) signal, the relative EMG signal, and the flexion relaxation ratio. RESULTS. Disability and pain scores improved significantly after the 12-week intervention. There were no changes in the active components of the flexion relaxation measurement but an approximate 67% decrease in the amount of activity measured during the relaxation phase at full trunk flexion. CONCLUSION. The data suggest that afferent feedback changes may be explaining why there is improved electrical relaxation following an exercise intervention. .COPYRG. 2006 Lippincott Williams & Wilkins, Inc.

L2 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1993:532618 CAPLUS <<LOGINID::20080104>>

DN 119:132618

TI Determination of pig apolipoprotein B genotype by gene amplification and restriction fragment length polymorphism analysis

AU Kaiser, M. E.; Nevin, D. N.; Sturley, S. L.; Purtell, C.; Attie, A. D.

CS Dep. Comp. Biosci., Univ. Wisconsin, Madison, WI, 53706, USA

SO Animal Genetics (1993), 24(2), 117-20

CODEN: ANGE3; ISSN: 0268-9146

DT Journal

LA English

AB Sequence differences within the pig apolipoprotein B (apoB) gene can be used to identify rapidly four of eight known pig apoB alleles, designated LBP1-LBP8. The use of gene amplification, followed by endonuclease digestion and agarose gel electrophoresis, to discern size and restriction site differences is described. LBP5, a common allele assocd. with reduced low d. lipoprotein clearance and hypercholesterolemia in pigs, is identified by a 283-bp insertion in intron 28. LBP3 and ***LBP7*** are distinguished by a unique HindIII site; LBP8 shares a unique HincII site with LBP5. This method facilitates identification of the apoB genotype of pigs used in lipoprotein research and allows for further investigation into the assocn. of particular apoB alleles with lipoprotein metab. abnormalities.

=> s T22G5.2

L3 1 T22G5.2

=> d bib abs

L3 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2003:6104 CAPLUS <<LOGINID::20080104>>

DN 138:67843

TI Methods of assaying genes of C. elegans involved in adult lifespan regulation and uses for gene therapy

IN Kenyon, Cynthia; Apfeld, Javier; Dillin, Andrew; Garigan, Delia; Hsu, Ao-Lin A.; Lehrer-Graier, Josh; Murphy, Coleen

PA The Regents of the University of California, USA

SO PCT Int. Appl., 112 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2003000861 A2 20030103 WO 2002-US20247 20020624
WO 2003000861 A9 20030410
WO 2003000861 A3 20030710

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MY, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW

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CA 2451247 A1 20030103 CA 2002-2451247 20020624
AU 2002350597 A1 20030108 AU 2002-350597 20020624
US 2003190312 A1 20031009 US 2002-179766 20020624

EP 1406489 A2 20040414 EP 2002-752095 20020624
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US 2006162002 A1 20060720 US 2005-282262 20051118
PRAI US 2001-300577P P 20010622

US 2001-301052P P 20010625
US 2002-373975P P 20020418
US 2002-179766 A1 20020624

WO 2002-US20247 W 20020624

AB The present invention is directed to methods of assaying for genes, gene products, and genes in pathways controlled by such genes and gene products, using RNAi and microarray anal., that regulate lifespan (e.g., extend or truncate adult lifespan) in eukaryotes such as invertebrates (e.g., C. elegans), plants, and mammals, e.g., humans. For example, the present invention is directed to genes encoding components of the mitochondrial respiratory chain and genes encoding glycolysis enzymes, which are involved in lifespan regulation, and genes and gene products in pathways controlled by such genes. Other genes and gene products identified as regulating aging and aging pathways include a gene encoding a GTPase; a transcriptional activator; novel genes: llw-1, llw-2, llw-3, and llw-4; genes encoding cytochrome P 450 proteins (involved in steroid biosynthesis); a melatonin synthesis gene; genes encoding insulin and insulin-like peptides; genes encoding heat shock factors; genes encoding catalases; stress-response genes; and metabolic genes. The invention further relates to methods for identifying and using agents, including small mol. chem. compns., antibodies, antisense nucleic acids, and ribozymes, that regulate, e.g., enhance, adult lifespan via modulation of aging assocd. proteins; as well as to the use of expression profiles, markers, and compns. in diagnosis and therapy related to lifespan extension, life expectancy, and aging. The present invention also relates to gene therapy involving lifespan assocd. genes.

=> s age or aging or lifespan

L4 2626198 AGE OR AGING OR LIFESPAN

=> s l4 and regula?

L5 149161 L4 AND REGULA?

=> s l5 and review

L6 13428 L5 AND REVIEW

=> d bib abs 1-10

L6 ANSWER 1 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson

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STN

AN 2008:49198 BIOSIS <<LOGINID::20080104>>

DN PREV200800045245

TI Kinetics, role and therapeutic implications of endogenous soluble form of receptor for advanced glycation end products (sRAGE) in diabetes.

AU Yamagishi, Sho-ichi [Reprint Author]; Matsui, Takanori; Nakamura, Kazuo

CS Kurume Univ, Sch Med, Dept Med, Div Cardiovasc Med, Kurume, Fukuoka

8300011, Japan

shoichi@med.kurume-u.ac.jp

SO Current Drug Targets, (OCT 2007) Vol. 8, No. 10, pp. 1138-1143.

ISSN: 1389-4501.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 4 Jan 2008

Last Updated on STN: 4 Jan 2008

AB Reducing sugars can react non-enzymatically with amino groups of protein to form Amadori products. These early glycation products undergo further complex reaction such as rearrangement, dehydration, and condensation to become irreversibly cross-linked, heterogeneous fluorescent derivatives, termed advanced glycation end products (AGEs). The formation and accumulation of AGEs have been known to progress at an accelerated rate in diabetes. There is a growing body of evidence that AGEs and their receptor (RAGE) axis is implicated in the pathogenesis of diabetic vascular complications. Indeed, the engagement of RAGE with AGEs is shown to elicit oxidative stress generation and subsequently evoke inflammatory responses in various types of cells, thus playing an important role in the development and progression of diabetic micro- and macroangiopathy. Moreover, administration of a recombinant soluble form of RAGE (sRAGE), has been shown to suppress the development of accelerated atherosclerosis

in diabetic apolipoprotein E-null mice. These observations suggest that exogenously administered sRAGE may capture and eliminate circulating AGEs, thus protecting against the AGEs-elicited tissue damage by acting as a decoy receptor. Recently, endogenous sRAGE has been identified in humans. However, there is few comprehensive ***review*** about the ***regulation*** and role of endogenous sRAGE in diabetes. In the former part of this paper, we ***review*** the role of the ***AGE***-RAGE system in the pathogenesis of diabetic vascular complications. Then we summarize in the latter part of this ***review*** the kinetics and pathophysiological role of endogenous sRAGE in diabetes. We also discuss the possibility that endogenous sRAGE may be a therapeutic target for the prevention of diabetic vascular complications.

L6 ANSWER 2 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2008:46781 BIOSIS <<LOGINID::20080104>>

DN PREV200800045726

TI Quality-of-life in elderly patients with cancer: A short ***review***

AU Wedding, Ulrich [Reprint Author]; Pienika, Ludger; Hoeffken, Klaus

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SO European Journal of Cancer, (OCT 2007) Vol. 43, No. 15, pp. 2203-2210.

CODEN: EJCAEL. ISSN: 0959-8049.

DT Article

LA English

ED Entered STN: 4 Jan 2008

Last Updated on STN: 4 Jan 2008

AB Background: Prolongation of survival and maintenance or improvement of health-related quality-of-life (HRQoL) are the two important goals within the treatment of individual patients. Due to the severity of symptoms and the toxicity of treatment, HRQoL has become a major area of concern when treating cancer patients in general and elderly patients in particular. Patients and methods: We present a literature ***review*** of HRQoL aspects in elderly patients with cancer and especially address the topic whether impairments in the different tools of a comprehensive geriatric assessment (CGA) are associated with decreased HRQoL in elderly cancer patients. Results: Elderly cancer patients tend to weight their HRQoL as more important than gain in survival, when compared to younger patients. An ***age***-dependent decrease in different scales of HRQoL is reported in patients and normative samples. HRQoL is also a predictor of survival. The variation of HRQoL can be used in trials comparing different treatment options. In individual patients, ***regular*** measurement of HRQoL aims to improve patients-centred care. ***Age*** related impairments of different areas of CGA are associated with decreased HRQoL in elderly cancer patients. Conclusions: HRQoL is an important outcome with elderly cancer patients and should be assessed ***regularly*** and thoroughly. (C) 2007 Elsevier Ltd. All rights reserved.

L6 ANSWER 3 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2008:42460 BIOSIS <<LOGINID::20080104>>

DN PREV200800036414

TI Telomere length profiles in humans - All ends are not equal.

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SO Cell Cycle, (OCT 15 2007) Vol. 6, No. 20, pp. 2486-2494.

ISSN: 1538-4101. E-ISSN: 1551-4005.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 27 Dec 2007

Last Updated on STN: 27 Dec 2007

AB Telomere length is an important parameter of telomere function since it determines number of aspects controlling chromosome stability and cell division. Since telomeres shorten with ***age*** in humans and premature ***aging*** syndromes are often associated with the presence of short telomeres, it has been proposed that telomere length is also an important parameter for organismal ***aging***. How mean telomere lengths are determined in humans remains puzzling, but it is clear that genetic and epigenetic factors appear to be of great importance. Experimental evidence obtained from many different organisms has provided the basis for a widely accepted counting mechanism based on a negative feedback loop for telomerase activity at the level of individual telomeres. In addition, recent studies in both normal and pathological contexts point to the existence of chromosome-specific mechanisms of telomere length ***regulation*** determining a telomere length profile, which is inherited and maintained throughout life. In this ***review***, we recapitulate the available data, propose a synthetic view of telomere length control mechanisms in humans and suggest new approaches to test current hypotheses.

L6 ANSWER 4 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2008:31851 BIOSIS <<LOGINID::20080104>>

DN PREV200800029157

TI Adipokine gene expression in brain and pituitary gland.

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SO Neuroendocrinology, (2007) Vol. 86, No. 3, pp. 191-209.

CODEN: NUNDAJ. ISSN: 0028-3835.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 19 Dec 2007

Last Updated on STN: 19 Dec 2007

AB The brain has been recognized as a prominent site of peptide biosynthesis for more than 30 years, and many neuropeptides are now known to be common to gut and brain. With these precedents in mind it is remarkable that adiposederived peptides like leptin have attracted minimal attention as brain-derived putative neuromodulators of energy balance. This ***review*** outlines the evidence that several adipose-specific genes are also expressed in the central nervous system and pituitary gland. We, and others, confirmed that the genes for leptin, resistin, adiponectin, FIAF (fasting-induced adipose factor) and adiponutrin are expressed and ***regulated*** in these tissues. For example, leptin mRNA was readily detectable in human, rat, sheep and pig brain, but not in the mouse. Leptin expression in rat brain and pituitary was ***regulated*** through development, by food restriction, and following traumatic brain injury. In contrast, hypothalamic resistin mRNA was unaffected by ***age*** or by fasting, but was significantly depleted by food restriction in mouse pituitary gland. Similar results were seen in the ob/ob mouse, and we noted a marked reduction in resistin-positive hypothalamic nerve fibres. Resistin and fiaf mRNA were also upregulated in hypoxic/schaemic mouse brain. Our studies on the ***regulation*** of neuronal adipokines were greatly aided by the availability of clonal hypothalamic neuronal cell lines. One of these, N-1, expresses both rsin and fiaf together with several other neuropeptides and receptors involved in energy homeostasis. Selective silencing of rsin revealed an autocrine/paracrine ***regulatory*** system, mediated through socs-3 expression that may influence the feedback effects of insulin and leptin in vivo. A similar convergence of signals in the pituitary gland could also influence anterior pituitary hormone secretion. In conclusion, the evidence is suggestive that brain and pituitary-derived adipokines represent a local ***regulatory*** circuit that may fine tune the feedback effects of adipose hormones in the control of energy balance. Copyright (C) 2007 S. Karger AG, Basel

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AN 2008:27148 BIOSIS <<LOGINID::20080104>>

DN PREV200800017125

TI Methionine sulfoxide reductases: selenoprotein forms and roles in antioxidant protein repair in mammals.

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SO Biochemical Journal, (NOV 1 2007) Vol. 407, No. Part 3, pp. 321-329.

ISSN: 0264-6021. E-ISSN: 1470-8728.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 19 Dec 2007

Last Updated on STN: 19 Dec 2007

AB Msrs (methionine sulfoxide reductases), MsrA and MsrB, are repair enzymes that reduce methionine sulfoxide residues in oxidatively damaged proteins to methionine residues in a stereospecific manner. These enzymes protect cells from oxidative stress and have been implicated in delaying the ***aging*** process and progression of neurodegenerative diseases. In recent years, significant efforts have been made to explore the catalytic properties and physiological functions of these enzymes. In the current ***review***, we present recent progress in this area, with the focus on mammalian MsrA and MsrBs including their roles in disease, evolution and function of selenoprotein forms of MsrA and MsrB, and the biochemistry of these enzymes.

L6 ANSWER 6 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2008:25935 BIOSIS <<LOGINID::20080104>>

DN PREV200800029314

TI Association between genetic variants in sortilin-related receptor 1

(SORL1) and Alzheimer's disease in adults with Down syndrome.

AU Lee, Joseph. H.; Chulikavit, Manut; Pang, Deborah; Zigman, Warren B.;

Silverman, Wayne; Schupf, Nicole [Reprint Author]

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SO Neuroscience Letters, (SEP 25 2007) Vol. 425, No. 2, pp. 105-109.

CODEN: NELEDS. ISSN: 0304-3940.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 19 Dec 2007

Last Updated on STN: 19 Dec 2007

AB Recent reports have suggested that variants in the sortilin-related

receptor gene (SORL1) increase the risk of late onset Alzheimer's disease (AD) in Northern European, Hispanic, African-American and Israeli-Arab populations. SORL1 directs trafficking of amyloid precursor protein (APP) and under-expression of SORL1 may lead to over-expression of P amyloid peptides. Adults with Down syndrome (DS) over-express APP and have early onset and high risk for AD. We investigated the relation of seven variants in the gene for SORL1 to ***age*** at onset and risk for AD among 208 adults with DS, 45-70 years of ***age*** at baseline. Participants were ascertained through the New York State developmental disability service system and followed at 18-month intervals. Information from cognitive assessments, caregiver interviews, medical record ***review*** and neurological examination was used to establish the diagnosis of dementia. Homozygosity for the minor T allele in rs556349 and for the minor C allele in rs536360 was associated with later ***age*** at onset and reduced risk of AD (HR = 0.26, 95% CI: 0.08-0.86; and HR = 0.40, 95% CI: 0.16-0.98, respectively). Mean ***age*** at onset was approximately four years later in individuals who were homozygous for those alleles compared with those who had at least one major allele. These findings indicate a modest association of variants in SORL1 with AD. In addition, we did not observe the same alleles to be associated with AD compared with earlier studies, suggesting that these SNPs are in linkage disequilibrium (LD) with the putative functional variants or that expression of the SORL1 gene and hence its interaction with APP might be modified by the extremely high levels of APP characteristic of Down syndrome. Thus, further studies are needed to identify functional variants that influence risk for AD in this uniquely vulnerable population. (c) 2007 Elsevier Ireland Ltd. All rights reserved.

L6 ANSWER 7 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
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AN 2008:24407 BIOSIS <<LOGINID::20080104>>

DN PREV200800031491

TI Fluid and electrolyte homeostasis in healthy subjects during prolonged hypokinesia.

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SO Trace Elements and Electrolytes, (2007) Vol. 24, No. 4, pp. 193-201. ISSN: 0946-2104.

DT Article

LA English

ED Entered STN: 19 Dec 2007

Last Updated on STN: 19 Dec 2007

AB Hypokinesia (diminished movement) is a factor of catabolism induction which may be developed, whenever there is a significant reduction of physical load on skeletal muscle system. Hypokinesia (HK) may occur due to ***age***, disease, disability and sedentary living and working conditions. Among the effects of HK, the changes in fluid and electrolytes (sodium, chloride, potassium, phosphorus, calcium and magnesium) homeostasis and in particular in fluid and electrolyte deposition (ability of the body to use electrolytes and fluid after all fluid and electrolyte loss have been established) have been of the greatest interest. This is due to the higher fluid and electrolyte loss with higher than lower fluid and electrolyte depletion and the recent interest on fluid and electrolyte homeostasis and fluid and electrolyte deposition under sedentary living and working conditions. Stable volume, osmotic concentration and electrolyte composition of internal fluid is one of the mandatory prerequisites for humans to maintain adequate physiological and biochemical condition and a highly efficient physical state. However, physiological and biochemical systems that ***regulate*** the concentration of each electrolyte in blood and other endogenous fluids, and the balance between the consumption and elimination of electrolytes from the body and thus total electrolyte content of the body are drastically affected. At present some information has been accumulated about the effect of prolonged HK on fluid and electrolyte homeostasis and fluid and electrolyte loss with fluid and electrolyte depletion. To this end, the aim of this ***review*** was to report the findings on the fluid and electrolyte homeostasis and fluid-electrolyte loss with whole body fluid and electrolyte depletion.

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AN 2008:13833 BIOSIS <<LOGINID::20080104>>

DN PREV20080006313

TI Mood disorders and fertility in women: a critical ***review*** of the literature and implications for future research.

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CS Stanford Univ, Dept Psychiat and Behav Sci, Stanford Ctr Neurosci Womenh Helath, Stanford, CA 94305 USA
elliew@stanford.edu

SO Human Reproduction Update, (NOV-DEC 2007) Vol. 13, No. 6, pp. 607-616. ISSN: 1355-4786.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 12 Dec 2007

Last Updated on STN: 27 Dec 2007

AB A medline literature ***review*** of fertility and mood disorder articles published since 1980 was performed in order to critically

review the literature regarding a relationship between mood disorders, fertility and infertility treatment. Previous studies suggests that mood disorders, both in the bipolar and unipolar spectrum, may be associated with decreased fertility rates. Most studies report that women seeking treatment for infertility have an increased rate of depressive symptoms and possibly major depression (none showed evaluated mood elevations). Many, but not all, studies found that depressive symptoms may decrease the success rate of fertility treatment. Treatments for infertility may independently influence mood through their effects on estrogen and progesterone, which have been shown to influence mood through their actions on serotonin. Studies are limited in scope and confounding variables are many, limiting the strength of the results. In conclusion, a range of existing studies suggests that fertility and mood disorders are related in a complex way. Future studies should use clinical interviews and standardized and validated measures to confirm the diagnosis of mood disorders and control for the variables of medication treatment, desire for children, frequency of sexual intercourse, ***age***, FSH levels, menstrual cycle ***regularity*** in assessing an interrelationship between mood disorders and fertility.

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AN 2008:12030 BIOSIS <<LOGINID::20080104>>

DN PREV20080005089

TI Long-chain polyunsaturated fatty acids and the ***regulation*** of bone metabolism.

AU Poulsen, Raewyn C. [Reprint Author]; Moughan, Paul J.; Kruger, Marlena C.

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SO Experimental Biology and Medicine (Maywood), (NOV 2007) Vol. 232, No. 10, pp. 1275-1288.

ISSN: 1535-3702.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 12 Dec 2007

Last Updated on STN: 12 Dec 2007

AB The role of prostaglandin E2 (PGE2) in the ***regulation*** of bone remodeling is well established. There is increasing evidence that various long-chain polyunsaturated fatty acids (LCPUFAs), as well as nonprostanoid LCPUFA metabolites, also have critical roles in ***regulating*** bone metabolism and may have therapeutic potential in the management of postmenopausal osteoporosis. Although only the 18-carbon precursors for the n-3 and n-6 LCPUFAs are deemed "dietary essential," the ability of the body to convert these precursor fatty acids into the more highly unsaturated 20- and 22-carbon LCPUFAs decreases with ***aging***, menopause, and various lifestyle factors (e.g., smoking). Increasing dietary LCPUFA intake increases tissue and blood LCPUFA concentrations, as well as the concentrations of their metabolites. Modification of dietary LCPUFA content, particularly increasing the intake of n-3 LCPUFAs, has been shown to minimize the decline in bone mass caused by menopause in women and ovariectomy in animal models. This ***review*** summarizes findings from both in vivo and in vitro studies and outlines the effects of LCPUFAs and their metabolites on calcium balance, osteoblastogenesis, osteoclastogenesis, and osteoblast and osteoclast function.

L6 ANSWER 10 OF 13428 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
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AN 2008:11678 BIOSIS <<LOGINID::20080104>>

DN PREV20080004301

TI Controlling mutation: intervening in evolution as a therapeutic strategy.

AU Cirz, Ryan T.; Romesberg, Floyd E. [Reprint Author]

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SO Critical Reviews in Biochemistry and Molecular Biology, (2007) Vol. 42, No. 5, pp. 341-354.

CODEN: CRBBEJ. ISSN: 1040-9238.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 12 Dec 2007

Last Updated on STN: 12 Dec 2007

AB Mutation is the driving force behind many processes linked to human disease, including cancer, ***aging***, and the evolution of drug resistance. Mutations have traditionally been considered the inevitable consequence of replicating large genomes with polymerases of finite fidelity. Observations over the past several decades, however, have led to a new perspective on the process of mutagenesis. It has become clear that, under some circumstances, mutagenesis is a ***regulated*** process that requires the induction of pro-mutagenic enzymes and that, at least in bacteria, this induction may facilitate evolution. Herein, we ***review*** what is known about induced mutagenesis in bacteria as well as evidence that it contributes to the evolution of antibiotic resistance. Finally, we discuss the possibility that components of induced mutation pathways might be targeted for inhibition as a novel therapeutic strategy to prevent the evolution of antibiotic resistance.

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FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 18:48:06 ON 04 JAN 2008

L1 4 S LBP 7 OR LBP7
L2 4 DUP REM L1 (0 DUPLICATES REMOVED)
L3 1 S T22G5.2
L4 2626198 S AGE OR AGING OR LIFESPAN
L5 149161 S L4 AND REGULA?
L6 13428 S L5 AND REVIEW

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L7 5534 L4 (3A) REGULAT?

=> s l7 and review
L8 929 L7 AND REVIEW

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L8 ANSWER 1 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
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AN 2008:49198 BIOSIS <<LOGINID::20080104>>
DN PREV200800045245

TI Kinetics, role and therapeutic implications of endogenous soluble form of receptor for advanced glycation end products (sRAGE) in diabetes.

AU Yamagishi, Sho-ichi [Reprint Author]; Matsui, Takanori; Nakamura, Kazuo
CS Kurume Univ, Sch Med, Dept Med, Div Cardiovasc Med, Kurume, Fukuoka 8300011, Japan
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SO Current Drug Targets, (OCT 2007) Vol. 8, No. 10, pp. 1138-1143.
ISSN: 1389-4501.

DT Article
General Review; (Literature Review)

LA English

ED Entered STN: 4 Jan 2008

Last Updated on STN: 4 Jan 2008

AB Reducing sugars can react non-enzymatically with amino groups of protein to form Amadori products. These early glycation products undergo further complex reaction such as rearrangement, dehydration, and condensation to become irreversibly cross-linked, heterogeneous fluorescent derivatives, termed advanced glycation end products (AGEs). The formation and accumulation of AGEs have been known to progress at an accelerated rate in diabetes. There is a growing body of evidence that AGEs and their receptor (RAGE) axis is implicated in the pathogenesis of diabetic vascular complications. Indeed, the engagement of RAGE with AGEs is shown to elicit oxidative stress generation and subsequently evoke inflammatory responses in various types of cells, thus playing an important role in the development and progression of diabetic micro- and macroangiopathy. Moreover, administration of a recombinant soluble form of RAGE (sRAGE), has been shown to suppress the development of accelerated atherosclerosis in diabetic apolipoprotein E-null mice. These observations suggest that exogenously administered sRAGE may capture and eliminate circulating AGEs, thus protecting against the AGEs-elicited tissue damage by acting as a decoy receptor. Recently, endogenous sRAGE has been identified in humans. However, there is few comprehensive ***review*** about the regulation and role of endogenous sRAGE in diabetes. In the former part of this paper, we ***review*** the role of the AGE-RAGE system in the pathogenesis of diabetic vascular complications. Then we summarize in the latter part of this ***review*** the kinetics and pathophysiological role of endogenous sRAGE in diabetes. We also discuss the possibility that endogenous sRAGE may be a therapeutic target for the prevention of diabetic vascular complications.

L8 ANSWER 2 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
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AN 2007:528569 BIOSIS <<LOGINID::20080104>>
DN PREV200700519859

TI Healthy ***aging*** : ***regulation*** of the metabolome by cellular redox modulation and prooxidant signaling systems: The essential roles of superoxide anion and hydrogen peroxide.

AU Linnane, Anthony William [Reprint Author]; Kios, Michael; Vitetta, Luis
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SO Biogerontology, (OCT 2007) Vol. 8, No. 5, pp. 445-467.
ISSN: 1389-5729.

DT Article
General Review; (Literature Review)

LA English

ED Entered STN: 10 Oct 2007

Last Updated on STN: 21 Nov 2007

AB The production of reactive oxygen species (ROS) and reactive nitrogen species (RNS) has long been proposed as leading to random deleterious modification of macromolecules with an associated progressive development of age associated systemic disease. ROS and RNS formation has been posited as a major contributor to the aging process. On the contrary, this ***review*** presents evidence that superoxide anion (and hydrogen peroxide) and nitric oxide (and peroxynitrite) constitute regulated prooxidant second messenger systems, with specific sub-cellular locales of production and are essential for normal metabolome and physiological function. The role of these second messengers in the regulation of the metabolome is discussed in terms of radical formation as

an essential contributor to the physiologically normal regulation of sub-cellular bioenergy systems; proteolysis regulation; transcription activation; enzyme activation; mitochondrial DNA changes; redox regulation of metabolism and cell differentiation; the concept that orally administered small molecule antioxidant therapy is a chimera. The formation of superoxide anion/hydrogen peroxide and nitric oxide do not conditionally lead to random macromolecular damage; under normal physiological conditions their production is actually regulated consistent with their second messenger roles.

L8 ANSWER 3 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
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AN 2007:518754 BIOSIS <<LOGINID::20080104>>
DN PREV200700526463

TI Aging of the mammalian circadian timing system: Changes in the central pacemaker and its regulation by photic and nonphotic signals.

AU Duncan, Marilyn J. [Reprint Author]

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SO Neuroembryology and Aging, (2006-2007) Vol. 4, No. 1-2, pp. 85-101.
ISSN: 1661-3406. E-ISSN: 1661-3414.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 10 Oct 2007

Last Updated on STN: 10 Oct 2007

AB Aging alters many aspects of endogenously regulated, 24-hour (circadian) rhythms, such as their amplitude, relationship to the ambient lighting cycle, and sensitivity to phase resetting signals. In order to elucidate the mechanisms responsible for these age-related changes, many studies have investigated age-related changes in the neural components of the circadian timing system, which include the hypothalamic suprachiasmatic nucleus (SCN), the site of the mammalian master circadian pacemaker, its afferent projections from the retina, thalamus, and midbrain raphe, and its efferent projections to the hypothalamus, thalamus and limbic system. Studies have shown that the SCN exhibits age-related changes in electrical activity rhythms in the absence of neurodegeneration. Also, aging selectively decreases SCN expression of the circadian clock genes, Clock and Bmal1, as well as vasoactive intestinal peptide. The latter participates in mediating light-induced phase resetting of the circadian pacemaker and the amplitude of the preovulatory luteinizing hormone surge, which are both attenuated by aging. Furthermore, by decreasing serotonin 5-HT7 receptors in the dorsal raphe, aging reduces one type of nonphotic input to the SCN. This ***review*** describes which aspects of circadian rhythm ***regulation*** exhibit changes during ***aging***, as well as which mechanisms appear to be spared or remain to be explored.

L8 ANSWER 4 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
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AN 2007:517678 BIOSIS <<LOGINID::20080104>>
DN PREV200700517861

TI Sirtuins: critical regulators at the crossroads between cancer and aging.

AU Saunders, L. R.; Verdin, E. [Reprint Author]

CS Univ Calif San Francisco, Gladstone Inst Virol and Immunol, 1650 Owens St, San Francisco, CA 94158 USA
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SO Oncogene, (AUG 13 2007) Vol. 26, No. 37, pp. 5489-5504.
CODEN: ONCNES. ISSN: 0950-9232.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 3 Oct 2007

Last Updated on STN: 3 Oct 2007

AB Sirtuins (SIRT1-7), or class III histone deacetylases (HDACs), are protein deacetylases/ADP ribosyltransferases that target a wide range of cellular proteins in the nucleus, cytoplasm, and mitochondria for post-translational modification by acetylation (SIRT1, -2, -3 and -5) or ADP ribosylation (SIRT4 and -6). The orthologs of sirtuins in lower organisms play a critical role in ***regulating*** ***lifespan***. As cancer is a disease of aging, we discuss the growing implications of the sirtuins in protecting against cancer development. Sirtuins regulate the cellular responses to stress and ensure that damaged DNA is not propagated and that mutations do not accumulate. SIRT1 also promotes replicative senescence under conditions of chronic stress. By participating in the stress response to genomic insults, sirtuins are thought to protect against cancer, but they are also emerging as direct participants in the growth of some cancers. Here, we ***review*** the growing implications of sirtuins both in cancer prevention and as specific and novel cancer therapeutic targets.

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AN 2007:437338 BIOSIS <<LOGINID::20080104>>
DN PREV200700441251

TI Interleukin-1 beta and caspase-1: Players in the ***regulation*** of ***age***-related cognitive dysfunction.

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SO Reviews in the Neurosciences, (2007) Vol. 18, No. 2, pp. 137-148.
ISSN: 0334-1763.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 15 Aug 2007

Last Updated on STN: 15 Aug 2007

AB Scientific research on the unprecedented and growing number of older adults in the United States and other industrialized countries has focused much attention on the health consequences of aging. Over the last few decades, inflammation in the brain and its implication in the progression of aging and age-related cognitive dysfunction has been an area of increasing importance to neuroscientists and is now considered as one of the most interesting and promising topics for aging research. One of the critical aspects of inflammatory processes is that the activation of one upstream inflammatory molecule initiates a cascade of self-sustaining inflammatory events which leads to the activation of a number of different downstream functions. Recently, a great deal of attention has been given to the interplay between inflammatory and apoptotic processes and the regulation of these processes by the caspases. The caspase family of proteases can be divided into proapoptotic and pro-inflammatory members. The present ***review*** summarizes recent observations of the interactions between the inflammatory cytokine interleukin-1 (IL-1) beta and the inflammatory/apoptotic caspase-1 and their involvement in age-related impairments in cognition. A comprehensive understanding of these mechanisms could potentially lead to the development of preventive or protective therapies that reduce or inhibit the cognitive decline associated with aging and age-related neurodegenerative disease.

L8 ANSWER 6 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
STN

AN 2007:382202 BIOSIS <<LOGINID::20080104>>

DN PREV200700376724

TI Basic mechanisms of the aging gastrointestinal tract.

AU Salles, N. [Reprint Author]

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nathalie.salles@chu-bordeaux.fr

SO Digestive Diseases, (2007) Vol. 25, No. 2, pp. 112-117.
ISSN: 0257-2753.

DT Article

LA English

ED Entered STN: 4 Jul 2007

Last Updated on STN: 4 Jul 2007

AB The goal of this short ***review*** is to summarize recent data on gastrointestinal changes with aging, focusing on gastrointestinal motility disorders, and mucosal variations. First of all, this ***review*** focused on gastrointestinal motility disorders with aging, even though an increased prevalence of several gastrointestinal motor disorders (i. e., dysphagia, dyspepsia, anorexia, and constipation) occurs in older people, aging per se appears to have a minor direct effect on most gastrointestinal functions. Secondly, this ***review*** focused on histological changes with ***aging***, i. e., ***regulation*** of gastrointestinal mucosal growth, gastrointestinal carcinogenesis, and gastric mucosal changes, especially changes in gastric acid secretion, bacterial overgrowth and its consequences on elderly patients. Copyright (C) 2007 S. Karger AG, Basel.

L8 ANSWER 7 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
STN

AN 2007:290236 BIOSIS <<LOGINID::20080104>>

DN PREV200700283088

TI SIRT1 in transcription regulation.

AU Luo Lan [Reprint Author]; Gao Zheng-Nan; Cheng Li-Jing; Yang Ze

CS Beijing Hosp, Inst Geriatr, Beijing 100730, Peoples R China

rolland_304224@sohu.com; yangze016@yahoo.com.cn

SO Zhongguo Shengwu Huaxue yu Fenzi Shengwu Xuebao, (MAR 20 2007) Vol. 23,

No. 3, pp. 187-193.

ISSN: 1007-7626.

DT Article

General Review; (Literature Review)

LA Chinese

ED Entered STN: 2 May 2007

Last Updated on STN: 24 Oct 2007

AB The silent mating type information regulation 1 (SIRT1) is a recently discovered NAD-dependent protein deacetylase. The SIRT1 sequence has the closest homology to the *S. cerevisiae* Sir2 protein. Conservation of SIRT1 regulation of the insulin-like growth factor I signaling pathway has been observed for *Caenorhabditis elegans* and mammals, indicating an ancient role for SIRT1 in modulation of organism adaptations to nutritional intake. SIRT1 regulates gene expression in a variety of organism by deacetylation of modified lysine residues on histones, transcription factors and other proteins. The human SIRT1 regulates a number of transcription factors that modulate metabolism and endocrine signaling, including peroxisome proliferator-activated receptor gamma, peroxisome proliferator-activated receptor gamma coactivator 1 alpha. Therefore SIRT1 is characteristically involved in the regulation of several crucial cellular pathways including different signal pathways to ***regulate*** mammalian ***lifespan***, gluconeogenesis and insulin

secretion and so forth, the transcription regulator may play potential role in clinical application and modern medicine. In this ***review***, the latest findings regarding SIRT1 gene and the role in regulation of transcription factors, mammalian lifespan and metabolism were reviewed and discussed.

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AN 2007:161576 BIOSIS <<LOGINID::20080104>>

DN PREV200700160349

TI Advances in endocrinology of aging research, 2005-2006.

AU Bellino, Francis L. [Reprint Author]

CS 4899 Elmer Derr Rd, Frederick, MD 21703 USA

bellinof@mail.nih.gov

SO Experimental Gerontology, (DEC 2006) Vol. 41, No. 12, pp. 1228-1233.

CODEN: EXGEAB. ISSN: 0531-5565.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 7 Mar 2007

Last Updated on STN: 24 Oct 2007

AB The purpose of this brief ***review*** is to highlight some of the more important advances in endocrinology of aging research over the past year. Four advances were chosen and briefly described. First, exploration of the early steps in the generation of the internal steroidal hormonal signal involved in lifespan extension via the insulin/IGF-like signaling pathway in the nematode by two research groups revealed that the product of cholesterol acid derivatives metabolized by a cytochrome P-450-like protein activates a protein with homology to the mammalian nuclear receptor superfamily, a process strikingly similar to the steroid hormone signaling pathway documented in mammalian systems. Second is the discovery that sirtuins, proteins that ***regulate*** ***lifespan*** in model organisms, enhance pancreatic insulin secretion in mice following a glucose challenge, suggesting the potential to ***regulate*** mammalian ***lifespan*** through ***regulation*** of the insulin signaling pathway. Third, the newly discovered hormone klotho, which also plays a role in ***regulating*** ***lifespan***, in this case in mice, is reported to not only negatively affect insulin sensitivity but, perhaps more importantly, significantly affects calcium and phosphate metabolism as a required cofactor of Fgf-23 signaling. Finally the gonadotropin FSH is shown to directly affect bone density in mice separate from any direct effect of estrogen, suggesting that reproductive hormones other than estrogen can directly impact menopause-associated pathophysiology in non-reproductive tissues. (c) 2006 Elsevier Inc. All rights reserved.

L8 ANSWER 9 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on
STN

AN 2007:114244 BIOSIS <<LOGINID::20080104>>

DN PREV200700112145

TI Vascular cell senescence - Contribution to atherosclerosis.

AU Minamino, Tohru; Komuro, Issei [Reprint Author]

CS Chiba Univ, Grad Sch Med, Dept Cardiovasc Sci and Med, Chuo Ku, 1-8-1

Inohana, Chiba 2608670, Japan

komuro-iky@umin.ac.jp

SO Circulation Research, (JAN 5 2007) Vol. 100, No. 1, pp. 15-26.

CODEN: CIRUAL. ISSN: 0009-7330.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 14 Feb 2007

Last Updated on STN: 14 Feb 2007

AB Cardiologists and most physicians believe that aging is an independent risk factor for human atherosclerosis, whereas atherosclerosis is thought to be a characteristic feature of aging in humans by many gerontologists. Because atherosclerosis is among the age-associated changes that almost always escape the influence of natural selection in humans, it might be reasonable to regard atherosclerosis as a feature of aging. Accordingly, when we investigate the pathogenesis of human atherosclerosis, it may be more important to answer the question of how we age than what specifically promotes atherosclerosis. Recently, genetic analyses using various animal models have identified molecules that are crucial for aging. These include components of the DNA-repair system, the tumor suppressor pathway, the telomere maintenance system, the insulin/Akt pathway, and other metabolic pathways. Interestingly, most of the molecules that influence the phenotypic changes of ***aging*** also ***regulate*** cellular senescence, suggesting a causative link between cellular senescence and aging. For example, DNA-repair defects can cause phenotypic changes that resemble premature aging, and senescent cells that show DNA damage accumulate in the elderly. Excessive calorie intake can cause diabetes and hyperinsulinemia, whereas dysregulation of the insulin pathway has been shown to induce cellular senescence in vitro. Calorie restriction or a reduction of insulin signals extends the lifespan of various species and decreases biomarkers of cellular senescence in vivo. There is emerging evidence that cellular senescence contributes to the pathogenesis of human atherosclerosis. Senescent vascular cells accumulate in human atheroma tissues and exhibit various features of dysfunction. In this ***review***, we examine the hypothesis that cellular senescence might contribute to atherosclerosis, which is a characteristic of aging in humans.

L8 ANSWER 10 OF 929 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on

STN

AN 2007:80046 BIOSIS <<LOGINID::20080104>>

DN PREV200700068923

TI A stress response pathway involving sirtuins, forkheads and 14-3-3 proteins.

AU Berdichevsky, Ala; Guarente, Leonard [Reprint Author]

CS MIT, Dept Biol, 31 Ames St, Cambridge, MA 02139 USA

leng@mit.edu

SO Cell Cycle, (NOV 15 2006) Vol. 5, No. 22, pp. 2588-2591.

ISSN: 1538-4101.

DT Article

LA English

ED Entered STN: 24 Jan 2007

Last Updated on STN: 24 Jan 2007

AB A conserved sir2 deacetylase gene can determine longevity of yeast, flies and worms. Recently we have reported a molecular mechanism of action of the C. elegans homologue sir-2.1. Our study revealed a novel stress-dependent pathway for lifespan determination in which SIR-2.1 binds to 14-3-3 proteins and a forkhead transcription factor DAF-16 to activate transcription of DAF-16 target genes. DAF-16 has long been known as a central protein in the ***regulation*** of ***lifespan*** that interfaces with multiple pathways. Recent studies by us and other laboratories suggest that DAF-16 requires co-factors for full activity. In this prospective we ***review*** recent literature highlighting the role of SIR-2.1, 14-3-3 and other DAF-16 co-factors in DAF-16 activation.

=> d his

(FILE 'HOME' ENTERED AT 18:47:57 ON 04 JAN 2008)

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 18:48:06 ON 04 JAN 2008

L1 4 S LBP 7 OR LBP7

L2 4 DUP REM L1 (0 DUPLICATES REMOVED)

L3 1 S T22G5.2

L4 2626198 S AGE OR AGING OR LIFESPAN

L5 149161 S L4 AND REGULA?

L6 13428 S L5 AND REVIEW

L7 5534 S L4 (3A) REGULAT?

L8 929 S L7 AND REVIEW

=> s l8 and lbp

L9 0 L8 AND LBP

=> s lbp and l4

L10 633 LBP AND L4

=> d bib abs

L10 ANSWER 1 OF 633 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on

STN

AN 2007:607075 BIOSIS <<LOGINID::20080104>>

DN PREV2007000612478

TI Fear avoidance beliefs do not influence disability and quality of life in Spanish elderly subjects with low back pain.

AU Kovacs, Francisco [Reprint Author]; Abaira, Victor; Cano, Alejandra;

Royuela, Ana; Gil del Real, Maria Teresa; Gestoso, Mario; Mufaggi,

Nicole; Muriel, Alfonso; Zamora, Javier

CS Fdn Kovacs, Dept Cientifico, Paseo Mallorca 36, E-07012 Palma de Mallorca, Spain

kovacs@kovacs.org

SO Spine, (SEP 1 2007) Vol. 32, No. 19, pp. 2133-2138.

CODEN: SPINDD. ISSN: 0362-2436.

DT Article

LA English

ED Entered STN: 6 Dec 2007

Last Updated on STN: 6 Dec 2007

AB Study Design. Correlation between previously validated questionnaires.Objective. To assess the association of fear avoidance beliefs (FAB) with disability and quality of life in elderly Spanish subjects.Summary of Background Data. As opposed to Anglo-Saxon and Northern European patients, in Spanish low back pain (***LBP***) patients of working ***age***, the influence of FAB on disability and quality of life is sparse and much less than that of pain. The influence of FAB on ***LBP*** -related disability and quality of life in the elderly is unknown.Methods. A visual analogue scale (VAS), the Roland Morris Questionnaire (RMQ), the FAB-Phys questionnaire (FABQ), and the SF-12 questionnaire were used to assess ***LBP***, disability, fear avoidance beliefs, and quality of life in 661 institutionalized elderly in Spain, 439 of whom had ***LBP***.Results. In all subjects, FAB correlated with ***LBP*** (r = 0.477), disability (r = 0.458), the Physical Component Summary of SF-12 (PCS SF-12) (r = -0.389), and the Mental Component Summary of SF-12 (MCS SF-12) (r = -0.165). In subjects with ***LBP***, FABs only correlated weakly with disability (r = -0.110). The stronger correlations were found between ***LBP*** and disability, both in all subjects (r = 0.803) and ***LBP*** patients (r = 0.445). Regression models including all the participants showed that the influence of FABs on physical quality of life was sparse and that FABs were not associated with either disability or mental quality of life. In elderly subjects with ***LBP***, FABs were not associated with either

disability or quality of life.Conclusion. In Spanish institutionalized elderly subjects, FABs only have a minor influence on physical quality of life, and none on disability or mental quality of life. In elderly subjects with ***LBP***, differences in FABs are not associated with differences in disability or quality of life. Further studies should explore the potential value of FABs in the elderly in other settings.

=> s lipid binding protein and l4

L11 65 LIPID BINDING PROTEIN AND L4

=> dup rem l11

PROCESSING COMPLETED FOR L11

L12 51 DUP REM L11 (14 DUPLICATES REMOVED)

=> d bib abs

L12 ANSWER 1 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2007:762489 CAPLUS <<LOGINID::20080104>>

DN 147:341248

TI Serum adipocyte fatty acid-binding protein levels were independently associated with carotid atherosclerosis

AU Yeung, D. C. Y.; Xu, A.; Cheung, C. W. S.; Wat, N. M. S.; Yau, M. H.; Fong, C. H. Y.; Chau, M. T.; Lam, K. S. L.

CS Department of Medicine and the Research Centre of Heart, Brain, Hormone, and Healthy Aging, Li Ka Shing Faculty of Medicine, Queen Mary Hospital, Univ. of Hong Kong, HKSAR, Peop. Rep. China

SO Arteriosclerosis, Thrombosis, and Vascular Biology (2007), 27(8), 1796-1802

CODEN: ATVBFA; ISSN: 1079-5642

PB Lippincott Williams & Wilkins

DT Journal

LA English

AB Objective: Adipocyte fatty acid-binding protein (A-FABP) has been shown to be an important player in atherosclerosis in animal models. However, the clin. relevance of these findings is still unknown. This study aims to examine the relationship between serum A-FABP level and carotid intima-media thickness (IMT), an indicator of atherosclerosis in humans. Methods and Results- The study cohort included 479 Chinese subjects who underwent carotid IMT measurement. Serum A-FABP levels were detd. by enzyme-linked immunosorbent assays. Serum A-FABP levels pos. correlated with carotid IMT in both men (r = 0.211, P = 0.001) and women (r = 0.435, P < 0.001). In women, but not in men, the presence of plaques was assocd. with significantly higher serum A-FABP levels (P < 0.001 vs. women without plaques). Stepwise multiple regression anal. showed that serum A-FABP level was independently assocd. with carotid IMT in women (P = 0.034), together with ***age*** and hypertension (both P < 0.001). Conclusions: A-FABP is an independent determinant of carotid atherosclerosis in Chinese women, but not in men. This gender difference may be attributed to the lower serum A-FABP levels in men, and the effect of other risk factors, such as smoking, among our male participants. Our results have provided clin. evidence supporting the role of A-FABP in the development of atherosclerosis.

RE.CNT 39 .THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d bib abs 2-10

L12 ANSWER 2 OF 51 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2007:442130 BIOSIS <<LOGINID::20080104>>

DN PREV200700428784

TI Reduced annexin II protein expression in high-grade prostatic intraepithelial neoplasia and prostate cancer.

AU Yee, David S.; Narula, Navneet; Ramzy, Ibrahim; Boker, John; Ahlering,

Thomas E.; Skarecky, Douglas W.; Ormstein, David K. [Reprint Author]

CS Univ Calif Irvine, Dept Urol, Med Ctr, 101 The City Dr,Bldg 55,Room 300,Rte 81, Orange, CA 92868 USA

domstei@uci.edu

SO Archives of Pathology & Laboratory Medicine, (JUN 2007) Vol. 131, No. 6, pp. 902-908.

CODEN: APLMAS. ISSN: 0003-9985.

DT Article

LA English

ED Entered STN: 15 Aug 2007

Last Updated on STN: 15 Aug 2007

AB Context. - Annexin II is a calcium-dependent phospho- ***lipid*** - ***binding*** ***protein*** that plays a role in many cellular functions, including apoptosis, signal transduction, and cellular motility. The protein is strongly expressed in normal prostatic epithelial glands, but its expression in benign prostatic lesions has not been reported. Although commonly underexpressed in prostate cancer, the association of reduced expression with pathologic grade and stage is unknown.Objective. - To compare annexin II expression in benign prostatic lesions with expression in high-grade prostatic intraepithelial neoplasia and prostate cancer, as well as to correlate expression levels with pathologic grade and stage.Design. - A semi-quantitative assessment of annexin II expression was performed in radical prostatectomy specimens from 74 patients and prostate needle core biopsy specimens from 13 patients. Foci with normal prostatic glands, atrophic glands, basal cell hyperplasia, high-grade prostatic intraepithelial neoplasia, and prostatic

adenocarcinoma were evaluated. Results. - Annexin II expression was present in more than 50% of glands in most (>85%) samples of benign prostatic epithelium, atrophic glands, and basal cell hyperplasia. In high-grade prostatic intraepithelial neoplasia, annexin II staining was markedly reduced in epithelial cells but not in basal cells. Annexin II was absent or focally present in moderately differentiated adenocarcinoma but was retained in poorly differentiated adenocarcinomas. Conclusions. - Reduced annexin II expression may be a useful diagnostic biomarker to help identify small foci of moderately differentiated adenocarcinoma on needle core biopsy specimens since it is consistently expressed in benign prostatic glands. Re-expression of annexin II in poorly differentiated adenocarcinoma may provide prognostic information.

L12 ANSWER 3 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:966059 CAPLUS <<LOGINID::20080104>>
DN 147:424776

TI Adipocyte fatty acid-binding protein is associated with markers of obesity, but is an unlikely link between obesity, insulin resistance, and hyperandrogenism in polycystic ovary syndrome women
AU Moehlig, Matthias; Weickert, Martin O.; Ghadami, Elham; Machlitt, Andrea; Pfeuffer, Bettina; Arfat, M.; Pfeiffer, Andreas F. H.; Schoell, Christof
CS Department of Endocrinology, Diabetes and Nutrition, Charite-University Medicine Berlin, Berlin, 12200, Germany
SO European Journal of Endocrinology (2007), 157(2), 195-200
CODEN: EJOEEP; ISSN: 0804-4643
PB BioScientifica Ltd.
DT Journal
LA English
AB Objective: Many polycystic ovary syndrome (PCOS) women suffer from adiposity and insulin resistance (IR), which play an important role in the development and maintenance of PCOS. Adipocyte fatty acid-binding protein (A-FABP) is mainly expressed in adipocytes, and circulating A-FABP has been associated with markers of obesity and IR. Thus, as observed with other adipose tissue derived factors, secreted A-FABP might be involved in the pathogenesis of obesity-associated disorders such as PCOS. Design: Plasma A-FABP concentrations were measured in 102 non-diabetic PCOS women, and associated

with markers of obesity, IR, inflammation, and hyperandrogenism were investigated by correlation and multiple linear regression analyses. The effect of lifestyle intervention on A-FABP was studied in a second cohort of 17 obese PCOS women. Results: A-FABP correlated with body mass index (BMI; $R = 0.694$, $P < 0.001$), dual-energy x-ray absorptiometry (DEXA) fat mass ($R = 0.729$, $P < 0.001$), DEXA lean body mass ($R = 0.399$, $P = 0.001$), HOMA %S ($R = -0.435$, $P < 0.001$), hsCRP ($R = 0.355$, $P = 0.001$), and free testosterone (fT; $R = 0.230$, $P = 0.02$). Adjusted for "age", smoking, and glucose metabolism, the association of A-FABP with HOMA %S was still significant ($P < 0.001$), whereas the associations with fT ($P = 0.09$) and hsCRP ($P = 0.25$) were not. Inclusion of BMI into the model abolished the impact of A-FABP on HOMA %S. In BMI-matched PCOS women ($n = 20$ pairs), neither HOMA %S ($P = 0.3$) nor fT ($P = 0.6$) were different despite different A-FABP levels ($P < 0.001$), and in 17 obese PCOS women undergoing a lifestyle intervention, changes in IR were not paralleled by changes in A-FABP. Conclusions: Circulating A-FABP was correlated with markers of obesity, but had no major impact on IR, inflammation, or hyperandrogenemia in PCOS women.

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:67089 CAPLUS <<LOGINID::20080104>>
DN 146:355212

TI Genetic variability affects the development of brown adipocytes in white fat but not in interscapular brown fat
AU Xue, Bingzhong; Rim, Jong-Seop; Hogan, Jessica C.; Coulter, Ann A.; Koza, Robert A.; Kozak, Leslie P.
CS Pennington Biomedical Research Center, Baton Rouge, LA, 70808, USA
SO Journal of Lipid Research (2007), 48(1), 41-51
CODEN: JLPRAW; ISSN: 0022-2275
PB American Society for Biochemistry and Molecular Biology, Inc.
DT Journal
LA English
AB Cold exposure induces brown adipocytes in retroperitoneal fat (RP) of adult A/J mice but not in C57BL/6J (B6) mice. In contrast, induction of the mitochondrial uncoupling protein 1 gene (Ucp1) in interscapular brown adipose tissue (iBAT) shows no strain dependence. We now show that unlike iBAT, in which Ucp1 was expressed in the fetus and continued throughout life, in RP, Ucp1 was transiently expressed between 10 and 30 days of "age" and then disappeared. Similar to the lack of genetic variation in the expression of Ucp1 in iBAT during cold induction of adult mice, no genetic variation in Ucp1 expression in iBAT was detected during development. In contrast, UCP1-positive multi-locular adipocytes, together with corresponding increases in Ucp1 expression, appeared in RP at 10 days of "age" in A/J and B6 mice, but with much higher expression in A/J mice. At 20 days of "age", brown adipocytes represent the major adipocyte present in RP of A/J mice. The disappearance of brown adipocytes by 30 days of "age" suggested that tissue remodeling occurred in RP. Genetic variability in Ucp1 expression could not be explained by variation in the expression of selective transcription factors and signaling molecules of adipogenesis. In summary, the existence of genetic variability between A/J and B6 mice during the development of brown adipocyte expression in RP, but not in iBAT, suggests that

developmental mechanisms for the brown adipocyte differentiation program are different in these adipose tissues.

RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:830502 CAPLUS <<LOGINID::20080104>>
DN 147:423577

TI Adipocyte- and Heart-Type Fatty Acid Binding Proteins Are Both Expressed in Subcutaneous and Intramuscular Porcine (Sus scrofa) Adipocytes
AU Gardan, Delphine; Louveau, Isabelle; Gondret, Florence
CS INRA, UMR1079 Systemes d'Elevage Nutrition Animale et Humaine, Saint Gilles, F-35590, Fr.
SO Comparative Biochemistry and Physiology, Part B: Biochemistry & Molecular Biology (2007), 148B(1), 14-19
CODEN: CBPBB8; ISSN: 1096-4959
PB Elsevier B.V.
DT Journal
LA English
AB Adipocyte- (A) and heart- (H) type fatty acid binding proteins (FABP) contribute to efficient fat storage and utilization, resp. To understand regional-differences in lipid metabolism between tissues, A- and H-FABP transcript and protein levels were studied in adipocytes isolated from s.c. adipose tissue or skeletal muscle in growing pigs (Sus scrofa). Interestingly, H-FABP was expressed in adipocytes isolated from both sites. We also showed that A-FABP and H-FABP were expressed at a lower level in i.m. adipocytes than in s.c. adipocytes. A discrepancy was observed between "age"-related changes in A-FABP content in isolated adipocytes and cell diameter or lipid content variations in tissues during growth.

RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 6 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:1012780 CAPLUS <<LOGINID::20080104>>
DN 145:349629

TI Glomerular and podocyte-specific expression profiling in mouse kidney and construction of the GlomChip microarray
IN Betsholtz, Christer; Tryggvason, Karl; Takemoto, Minoru; He, Liquan; Patrakkas, Jaakko
PA Swed.
SO PCT Int. Appl., 802pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006100066	A1	20060928	WO 2006-EP2646	20060322
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GR, GU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, GU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 2006216722	A1	20060928	US 2005-90997	20050325
PRAI US 2005-90997	A	20050325		

AB The present invention provides the assembly and use of a transcription-profiling platform dedicated to the study of mouse renal glomeruli. By using a novel method for glomerulus isolation, a series of high complexity EST libraries were constructed from glomeruli at different stages of development. From these libraries, a total of 15,627 EST clones were sequenced, and by annotation against ENSEMBL found to map to 6,053 different genes, estd. to cover 85% of the glomerular transcriptome. Microarray analysis of isolated glomeruli, non-glomerular kidney tissue, isolated extra-renal microvessel fragments, and FACS-sorted podocytes identified most known glomerular and podocyte-specific transcripts. To identify novel podocyte-specific transcripts, the EST clones were arrayed and hybridized against labeled targets from isolated glomeruli, non-glomerular kidney tissue, FACS-sorted podocytes, and brain capillary fragments. This revealed the existence of over 300 novel glomerular cell-enriched transcripts, the expression of many of which was further localized to podocytes, mesangial cells, and juxtaglomerular cells by *in situ* hybridization. For one of the podocyte-restricted transcripts, dendrin, previously regarded to be brain-specific, the protein was expressed, antibodies generated, and the antibodies used to localize dendrin to the podocyte foot processes. Quant. expression data are provided for known podocyte genes. Construction and performance of a glomerular transcript-specific microarray, designated GlomChip, is described.

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 7 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2006:33751 CAPLUS <<LOGINID::20080104>>

DN 144:128966

TI Constrained cyano compounds as selective inhibitors of dipeptidyl
peptidase IV, their preparation, pharmaceutical compositions, and use in
therapy

IN Campbell, David Alan; Betancort, Juan Manuel; Winn, David T.

PA USA

SO U.S. Pat. Appl. Publ., 35 pp.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2006009518	A1	20060112	US 2005-179797	20050712
CA 2573848	A1	20060216	CA 2005-2573848	20050712
WO 2006017292	A1	20060216	WO 2005-US24695	20050712
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
EP 1778220	A1	20070502	EP 2005-773420	20050712
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
PRAI US 2004-587391P	P	20040712		
WO 2005-US24695	W	20050712		
OS CASREACT 144:128966; MARPAT 144:128966				
GI				

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA
OFFLINE PRINT *

AB The invention relates to constrained cyano compds. of formula I, which are
selective inhibitors of dipeptidyl peptidase IV (DPP-IV). In compds. I, X
is (un)substituted C, optionally forming a double bond with one of the
carbon atoms to which it is attached, S, or O; R1 and R4 are independently
H, (un)substituted alkyl, (un)substituted alkenyl, (un)substituted
alkynyl, (un)substituted cycloalkyl, (un)substituted cycloalkyl-alkyl,
(un)substituted aryl, (un)substituted aralkyl, etc.; and R2, R3, R5, and
R6 are independently selected from H, F, Cl, Br, I, OH, NH2, CN, alkoxy,
(di)alkylamino, acyl, alkoxycarbonyl, aryloxy, etc. The invention also
relates to the prepn. of I, pharmaceutical compns. comprising a compd. I
together with at least one pharmaceutically acceptable carrier or diluent,
optionally in combination with another active ingredient, as well as to
the use of the compns. for treating, controlling, or preventing conditions
affected by dipeptidyl peptidase-IV inhibition. Esterification of
(S)-phenylglycine followed by condensation with benzaldehyde,
.alpha.-allylation, hydrolysis and N-protection gave amino acid II, which
underwent ozonolysis, ester hydrolysis, and cyclization with L-cysteine Me
ester to give thiazolidine III. Intramol. cyclocondensation of III,
amidation, dehydration and deprotection resulted in the formation of
hexahydropyridothiazole IV. The compds. of the invention are selective
for DPP-IV over other dipeptidyl peptidases with compd. IV being more than
100-fold selective for DPP-IV over DPP-VII, DPP-VIII, and fibroblast
activation protein (FAP) and between 10- and 100-fold for DPP-IV over
DPP-IX.

L12 ANSWER 8 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2006:741512 CAPLUS <<LOGINID::20080104>>

DN 145:245635

TI Klotho protein promotes adipocyte differentiation

AU Chihara, Yukana; Rakugi, Hiromi; Ishikawa, Kazuhiko; Ikushima, Masashi;
Maekawa, Yoshihiro; Ohta, Junsuken; Kida, Iwao; Ogiwara, Toshio

CS Department of Geriatric Medicine, Osaka University Graduate School of
Medicine, Osaka, 565-0871, Japan

SO Endocrinology (2006), 147(8), 3835-3842

CODEN: ENDOAO; ISSN: 0013-7227

PB Endocrine Society

DT Journal

LA English

AB Mice with homozygous disruption of the klotho exhibit multiple ***age***
related disorders and have barely detectable amts. of white adipose
tissue. Although klotho expression in cultured adipocytes has been
reported, little is known about its function in adipocytes. In the
present study, we investigated the role of klotho on adipocyte
differentiation. Adipocyte differentiation was induced by incubation of
confluent 3T3-L1 cells with insulin, dexamethasone, and
1-methyl-3-isobutyl-xanthine. Klotho-siRNA and expression vector were
produced for klotho suppression and overexpression, resp. Klotho protein
was purified for detn. of the hormonal effect of klotho. Klotho mRNA and
protein expression increased up to the 3rd d of differentiation. A
peroxisome proliferator-activated receptor-gamma. agonist increased

klotho expression during the early period of adipocyte differentiation.

The mRNA expression of adipocyte differentiation markers, such as
CCAAT/enhancer-binding protein (C/EBP).alpha., C/EBP.beta., C/EBP.delta.,
peroxisome proliferator-activated receptor-gamma., and fatty acid binding
protein 4, was decreased by klotho suppression, and increased 1.9- to
3.8-fold by klotho overexpression. The results of Oil Red O staining also
suggested that klotho overexpression promoted adipocyte differentiation.
Klotho protein stimulation resulted in a 2.4- to 4.6-fold increase in mRNA
expression of differentiation markers compared with control, and the time
course depended on adipocyte induction status. Western blot anal. showed
that protein levels of C/EBP.alpha. and C/EBP.delta. were increased by
Klotho protein stimulation. These results suggest that klotho works as a
hormonal factor to promote adipocyte differentiation in the early days,
during the period of transient proliferation in the differentiation
process, and that klotho may play an essential role in adipocyte
differentiation.

RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS
RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 9 OF 51 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2006:1070578 CAPLUS <<LOGINID::20080104>>

DN 145:349038

TI The farnesoid X receptor promotes adipocyte differentiation and regulates
adipose cell function in vivo

AU Rizzo, Giovanni; Disante, Moises; Mencarelli, Andrea; Renga, Barbara;
Gioglio, Animo; Pellicciari, Roberto; Fiorucci, Stefano

CS Dipartimento di Medicina Clinica e Sperimentale, University of Perugia,
Perugia, Italy

SO Molecular Pharmacology (2006), 70(4), 1164-1173

CODEN: MOPMA3; ISSN: 0026-895X

PB American Society for Pharmacology and Experimental Therapeutics

DT Journal

LA English

AB The differentiation of a preadipocyte into a mature adipocyte is a highly
regulated process that requires a scripted program of transcriptional
events leading to changes in gene expression. Several genes are assocd.
with adipogenesis, including the C/EBP/enhancer-binding protein (C/EBPs)
and peroxisome proliferator-activated receptor (PPAR) families of
transcription factors. In this study, we have investigated the role of
the farnesoid X receptor (FXR), a bile acid-activated nuclear receptor, in
regulating adipogenesis in a preadipocyte cell line (3T3-L1 cells). Our
results show that FXR is expressed in the white adipose tissue of adult
mice and in differentiated 3T3-L1 cells but not in undifferentiated
preadipocytes. Exposure of 3T3-L1 cells to INT-747 (6-EI
cheno-deoxycholic acid), a potent and selective FXR ligand, increases
preadipocyte differentiation induced by a differentiating mixt. contg.
insulin. Augmentation of differentiating mixt.-induced differentiation of
3T3-L1 cells by INT-747 assocd. with induction of aP2, C/EBP.alpha., and
PPAR.gamma.2 mRNAs along with other adipocyte-related genes. This effect
was reversed by guggulsterone, an FXR antagonist, and partially reverted
by GW9662 (2-chloro-5-nitro-N-phenylbenzamide), a selective PPAR.gamma.
antagonist, indicating that FXR modulates adipocyte-related genes by
PPAR.gamma.-dependent and -independent pathways. Regulation of
adipocyte-related genes by INT-747 was lost in FXR-/- mice, indicating
that modulation of these genes by INT-747 requires an intact FXR. In
addn., INT-747 enhances both insulin-induced serine phosphorylation of Akt
and glucose uptake by 3T3-L1 cells. Taken together, these results suggest
that activation of FXR plays a crit. role in regulating adipogenesis and
insulin signaling.

RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS
RECORD

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reserved on STN

AN 2006598155 EMBASE <<LOGINID::20080104>>

TI Localization of LRRK2 to membranous and vesicular structures in mammalian
brain.

AU Biskup S.; Moore D.J.; Celsi F.; Higashi S.; West A.B.; Andrabi S.A.;
Kurkinen K.; Yu S.-W.; Savitt J.M.; Waldevogel H.J.; Faull R.L.M.; Emson
P.C.; Torp R.; Ottersen O.P.; Dawson T.M.; Dawson V.L.

CS Dr. V.L. Dawson, Johns Hopkins University, School of Medicine, Broadway
Research Building, 733 North Broadway, Baltimore, MD 21205, United States.
vdawson@jhmi.edu

SO Annals of Neurology, (Nov 2006) Vol. 60, No. 5, pp. 557-569.

Refs: 23

ISSN: 0364-5134 CODEN: ANND3

CY United States

DT Journal; Article

FS 029 Clinical and Experimental Biochemistry

005 General Pathology and Pathological Anatomy

008 Neurology and Neurosurgery

LA English

SL English

ED Entered STN: 4 Jan 2007

Last Updated on STN: 4 Jan 2007

AB Objective: The PARK8 gene responsible for late-onset autosomal dominant
Parkinson's disease encodes a large novel protein of unknown biological
function termed leucine-rich repeat kinase 2 (LRRK2). The studies herein
explore the localization of LRRK2 in the mammalian brain. Methods:
Polyclonal antibodies generated against the amino or carboxy termini of
LRRK2 were used to examine the biochemical, subcellular, and

immunohistochemical distribution of LRRK2. Results: LRRK2 is detected in rat brain as an approximate 280kDa protein by Western blot analysis. Subcellular fractionation demonstrates the presence of LRRK2 in microsomal, synaptic vesicle-enriched and synaptosomal cytosolic fractions from rat brain, as well as the mitochondrial outer membrane. Immunohistochemical analysis of rat and human brain tissue and primary rat cortical neurons, with LRRK2-specific antibodies, shows widespread neuronal-specific labeling localized exclusively to punctate structures within perikarya, dendrites, and axons. Confocal colocalization analysis of primary cortical neurons shows partial yet significant overlap of LRRK2 immunoreactivity with markers specific for mitochondria and lysosomes. Furthermore, ultrastructural analysis in rodent basal ganglia detects LRRK2 immunoreactivity associated with membranous and vesicular intracellular structures, including lysosomes, endosomes, transport vesicles, and mitochondria. Interpretation: The association of LRRK2 with a variety of membrane and vesicular structures, membrane-bound organelles, and microtubules suggests an affinity of LRRK2 for lipids or lipid-associated proteins and may suggest a potential role in the biogenesis and/or regulation of vesicular and membranous intracellular structures within the mammalian brain. .COPYRG. 2006 American Neurological Association Published by Wiley-Liss, Inc., through Wiley Subscription Services.

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L13 3 A FABP AND (LIFESPAN OR AGING)

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YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L14 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:85781 CAPLUS <<LOGINID::20080104>>
DN 146:180307
TI Method for the determination of adipocyte fatty acid binding protein (
A - ***FABP*** , FABP4, P2) and diagnosis of metabolic syndrome,
diabetes type II and insulin resistance
PA Biovondor Laboratory Medicine, Inc., Czech Rep.
SO Ger. Offen., 36pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1
PATENT NO. KIND DATE APPLICATION NO. DATE
PI DE 102006034607 A1 20070125 DE 2006-102006034607 20060721
WO 2007063363 A2 20070607 WO 2006-1B2383 20060721
WO 2007063363 A3 20070830
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE,
GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR,
KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW,
MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC,
SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US,
UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
PRAI DE 2005-102005034788 IA 20050721

AB The invention concerns a method for diagnosing metabolic syndrome, its early forms and complications, diabetes type II, insulin resistance, obesity or similar diseases by detg. the concn. of adipocyte fatty acid binding protein ***A*** - ***FABP*** in a body fluid and using the result as an indicator of the fatty acid transport capacity. ***A*** - ***FABP*** is detd. by ELISA or similar dye-involving immunoassays in conjunction with microtiterplates; recombinant ***A*** - ***FABP*** is used as ref. material. For diagnosis addnl. data are taken into account, e.g. body mass index, glucose level.

L14 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:830502 CAPLUS <<LOGINID::20080104>>
DN 147:423577
TI Adipocyte- and Heart-Type Fatty Acid Binding Proteins Are Both Expressed in Subcutaneous and Intramuscular Porcine (Sus scrofa) Adipocytes
AU Gardan, Delphine; Louveau, Isabelle; Gondret, Florence
CS INRA, UMR1079 Systemes d'Elevage Nutrition Animale et Humaine, Saint Gilles, F-35590, Fr.
SO Comparative Biochemistry and Physiology, Part B: Biochemistry & Molecular Biology (2007), 148B(1), 14-19
CODEN: CBPBB8; ISSN: 1096-4959
PB Elsevier B.V.
DT Journal
LA English

AB Adipocyte- (A) and heart- (H) type fatty acid binding proteins (FABP) contribute to efficient fat storage and utilization, resp. To understand regional-differences in lipid metab. between tissues, A- and H-FABP transcript and protein levels were studied in adipocytes isolated from s.c. adipose tissue or skeletal muscle in growing pigs (Sus scrofa).

Interestingly, H-FABP was expressed in adipocytes isolated from both sites. We also showed that ***A*** - ***FABP*** and H-FABP were expressed at a lower level in i.m. adipocytes than in s.c. adipocytes. A discrepancy was obsd. between age-related changes in ***A*** - ***FABP*** content in isolated adipocytes and cell diam. or lipid content variations in tissues during growth.

RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L14 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:385929 CAPLUS <<LOGINID::20080104>>
DN 141:52311
TI Genetic polymorphisms affecting the phenotypic expression of familial hypercholesterolemia
AU Bertolini, Stefano; Pisciotto, Livia; Di Scala, Lilla; Langheim, Silvia; Bellocchio, Antonella; Masturzo, Paola; Cantafora, Alfredo; Martini, Scipione; Averna, Maurizio; Pes, Gianni; Stefanutti, Claudio; Calandra, Sebastiano
CS Department of Internal Medicine, University of Genoa, Genoa, I-16132, Italy
SO Atherosclerosis (Amsterdam, Netherlands) (2004), 174(1), 57-65
CODEN: ATHSBL; ISSN: 0021-9150
PB Elsevier
DT Journal
LA English
AB The clin. expression of heterozygous familial hypercholesterolemia (FH) is highly variable even in patients carrying the same LDL receptor (LDL-R) gene mutation. This variability might be due to environmental factors as well as to modifying genes affecting lipoprotein metab. We investigated Apo E (.epsilon.2, .epsilon.3, .epsilon.4), MTP (-493G/T), Apo B (-516C/T), Apo A-V (-1131T/C), HL (-514C/T and -250G/ ***A***), ***FABP*** -2 (A54T), LPL (D9N, N291S, S447X) and ABCA1 (R219K) polymorphisms in 221 unrelated FH index cases and 349 FH relatives with defined LDL-R gene mutations. We found a significant and independent effect of the following polymorphisms on: (i) plasma LDL-C (Apo E, MTP and Apo B); (ii) plasma HDL-C (HL, FABP-2 and LPL S447X); (iii) plasma triglycerides (Apo E and Apo A-V). In subjects with coronary artery disease (CAD+), the prevalence of FABP-2 54TT genotype was higher (16.5% vs. 5.2%) and that of ABCA1 219RK and KK genotypes lower (33.0% vs. 51.5%) than in subjects with no CAD. Independent predictors of increased risk of CAD were male sex, age, arterial hypertension, LDL-C level and FABP-2 54TT genotype, and of decreased risk the 219RK and KK genotypes of ABCA1. These findings show that several common genetic variants influence the lipid phenotype and the CAD risk in FH heterozygotes.

RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD

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